Social Science & Medicine 266 (2020) 113430

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: http://www.elsevier.com/locate/socscimed

Grow fast, die young? The causes and consequences of adult height and prolonged growth in nineteenth century Maastricht

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ARTICLE INFO

Keywords:
Height
Anthropometry
Growth
Mortality
Epidemiological transition
Netherlands
Nineteenth century
Historical demography

ABSTRACT

Background: Both adult body height and the developmental growth trajectory have been found to be important predictors of later-life mortality. However, evidence for these relationships largely comes from contemporary populations, where most people live until old-age. It is an open question how height and growth impact later-life mortality in a population where death before old-age is more common. We therefore study the causes and mortality consequences of height and growth in a high-mortality, nineteenth-century Dutch population.

Methods: We exploit a unique dataset from three sources: conscription records with late-adolescent height, standing militia registers with adult height, and individual cause-of-death and age-at-death information. Our study is set in the Dutch city of Maastricht. To determine the causes of height and growth (either early-life environmental conditions or shared family inheritance), we use Pearson's correlation tests and multilevel linear models. To determine height and growth's consequences, we use survival analyses, for all-cause mortality and cause-specific mortality.

Results: Regarding the causes of height and growth, we find that prolonged growth and adolescent height are more strongly associated with external environmental factors than shared family inheritance. Adult height is more strongly related to shared family inheritance. Regarding the consequences of height and growth, we find that being taller in adulthood and growing faster are significantly associated with an increased hazard of death for the all-cause mortality model.

Conclusions: While we find the 'usual suspects' for the causes of height and growth, our findings for the consequences are surprising: the tallest individuals who grow the fastest have the highest hazard of death. Our results may be explained by a selection effect: the tall, fast growers may be the least-selected in early-life, and are therefore more vulnerable than their peers in adulthood.

1. Introduction

A combination of adult height and the developmental growth trajectory may be important predictors of later-life mortality. Already, a number of studies have found that tallness is associated with decreased mortality risks (Waaler, 1984; Davey Smith et al., 2000). Another important component of body height is how fast, or at what age, terminal adult height is reached. There is evidence that growing later is the result of deprivation, and may incur a later-life health penalty (Metcalfe and Monaghan, 2001). To gain a fuller understanding of adult height and growth, we are interested in exploring both their causes and the consequences they may have for mortality.

Importantly, height and growth appear to vary by context. Epidemiological transition theory provides a helpful framework to understand how and why this is the case. Relative to pre-transition (or currently-transitioning) periods, post-transition periods see much longer life expectancies and fewer deaths from infectious diseases. This is largely due to improving environmental conditions, particularly those relating to food, public health and sanitation (Omran, 1971). A consequence of the epidemiological transition is that, over the past 150 years, people have gotten taller in much of the world (a vertical shift in height). At the same time, people began reaching adult height at earlier ages (a horizontal

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https://doi.org/10.1016/j.socscimed.2020.113430
Received in revised form 6 October 2020; Accepted 7 October 2020
Available online 10 October 2020

shift in time) (e.g. van Wieringen, 1972). Moreover, both growth velocity and duration - which we refer to collectively as growth trajectory - in pre-transition populations appear to have differed dramatically from today: slower growth over a longer period, and rapid growth after a period of deprivation (catch-up growth) were more common (Steckel, 2009; Beekink and Kok, 2017; Gao and Schneider, 2020).

The relationships between height, growth trajectory and mortality may also differ based on context. Regarding height's association with mortality, tallness is associated with living longer in post-transition populations (e.g. Waaler, 1984; Davey Smith et al., 2000). But studies conducted in historical, pre-transition contexts tended to find no, or even an inverted, relationship between adult height and mortality (for an extensive review, see Sear, 2010). Further, evidence for the negative consequences of more prolonged growth trajectories comes from contemporary, post-transition populations (Mangel and Munch, 2005). It remains an unanswered question if these negative mortality associations are borne out in pre-transition populations. In the present study, we are interested in understanding both the causes of prolonged growth trajectories and terminal adult height, and the consequences both may have had for mortality among adults in a pre-transition population.

1.1. Height as a measure of well-being

Adult height is considered to be an excellent index of population well-being. Average heights of a genetically 'closed' population are thought to represent shifts in net nutrition. This concept refers broadly to actual nutritional intake, minus energy lost to metabolic maintenance, physical activity and disease (Steckel, 2009). Changes in a population's average height roughly represent these various inputs across development, from conception through the terminus of puberty. Time series of average heights form an intuitively appealing and straightforward window into population well-being over time.

Adult height is not only reflective of good environmental conditions in early life; it is often predictive of well-being in later-life. Adult height, on both an individual and aggregate level, has been linked to beneficial later-life outcomes. These include socio-economic status and the likelihood of being married (Thompson et al., 2019; Yamamura and Tsutsui, 2017). However, height's influence on mortality is not clear: some studies found a link between height and later mortality, albeit with decreasing returns past a certain (tall) height (Waaler, 1984; Davey Smith et al., 2000). Other studies found the opposite, and instead argued that shortness is associated with later mortality (Samaras and Elrick, 1999; Weindruck and Sohal, 1997). As mentioned, studies in pre-transition populations generally did not find significant relationships between height and mortality (Sear, 2010).

1.2. The growth trajectory

Missing from this interpretation of adult height is the growth trajectory. Growth may be delayed in times of stresses on net nutrition until conditions improve, so that individuals' growth trajectories may vary over time (Oppers, 1963; Tanner, 1990). Existing research points to two types of prolonged growth trajectories: slower growth over a prolonged period of time; and a rapid increase in growth when conditions improve, termed catch-up growth (Boersma and Wit, 1997; Metcalfe and Monaghan, 2001). In historical contexts, both growth trajectories have been found (Beekink and Kok, 2017; Gao and Schneider, 2020). Consequently, and because we have two height measurements available and therefore cannot determine the shape of the growth curve, we do not disaggregate between these two types of prolonged growth trajectories.

A prolonged growth trajectory may explain why the relationship between terminal adult height and mortality may be absent or even inverted in pre-transition populations. As mentioned, due to more variable environmental conditions, prolonged growth trajectories are likely more prevalent in pre-transition populations. It may be that people who were subject to poor early-life conditions are nonetheless tall adults as a result of growing later and longer. Height is then less reflective of early-life conditions, and is therefore a more complicated indicator of later-life mortality.

Unfortunately, the biological mechanisms underpinning prolonged growth trajectories are not yet well-understood. Metcalfe and Monaghan (2013) found that prolonged growth may have short-term advantages, in particular, surviving until reproductive age. However, it may also strain the body, resulting in weaker organ function. Therefore, prolonged growers may be more likely to die earlier than their peers in adulthood (Metcalfe and Monaghan, 2013). The strongest evidence for this comes from the elevated risk of death from respiratory or cardiac diseases among those who experienced catch-up growth (Eriksson et al., 1999). Most studies demonstrating this relationship focused on critical periods in the first years of life. However, Lundberg (1993) argued for taking a longer period of possible deprivation into consideration, through the adolescent growth spurt. It is not clear if the period of deprivation is critical to determining the impact of growth trajectories (Kuh et al., 2004). Due to data constraints, we focus on growth between late adolescence and adulthood as a proxy for prolonged growth trajectory. Even with this different focus, we expect that the stress both of being deprived and short in early life, and of growing longer and later may result in earlier mortality.

How might these relationships be borne out in a pre-transition population? Two features of height in a pre-transition population may differently impact the height and growth trajectories' association with mortality. First, tallness may be directly penalized. Given that larger bodies and more growth require greater caloric intakes, a period of deprivation could more negatively impact taller individuals (Stulp and Barrett, 2016). Second, and in contrast, ultimately being taller, regardless of growth trajectory, may also indirectly increase longevity. On average, then as today, taller people earn more money, and attain higher occupational statuses (Thompson et al., 2020). Both height and occupational status are also inversely related to mortality (Alter et al., 2004). Moreover, although it is less common in historical populations, some studies point at a reversed J-shaped relation between adult height and mortality, with a height optimum between 185 and 190 centimeters (cm) (e.g. Waaler, 1984, for a contemporary population, and Costa, 2004, for a historical population).

Ultimately, we are interested in exploring both the causes and consequences of height and growth in a pre-transition population. We are, to our knowledge, the first to do either, and are certainly the first to do both. To accomplish this, we pose two questions: to what extent do environmental factors impact height and prolonged growth trajectories? and to what extent is the combination of adult height and prolonged growth associated with (cause-specific) mortality?

1.3. Maastricht in context

We focus on a late nineteenth-century cohort from the Dutch city of Maastricht, in the province of Limburg. The Dutch overall in this period typified a pre-transition population. Bonneux (2002) defines countries with an average male life expectancy of 50 years as high mortality, and uses the early-twentieth century Netherlands as an example. This is comparable to Lesotho today, which, with an average male life expectancy of 50, has the lowest life expectancy in the world (World Bank, 2020). In contrast, Bonneux (2002) defines the late twentieth century Netherlands as a low-mortality population, with an average life expectancy of 74 years for men in 1994 (and 80 years in 2017) (World Bank, 2020).

Late-nineteenth century Maastricht's living conditions were broadly poor, consistent and extreme. Whereas the majority of the Netherlands started their epidemiological transition in this period, Maastricht and the province of Limburg overall lagged behind. The infant mortality rate (IMR) illustrates this, because the youngest in a given population are affected most by poor living conditions (Fig. 1). In our observation period, the IMR in Maastricht remained persistently high relative to the

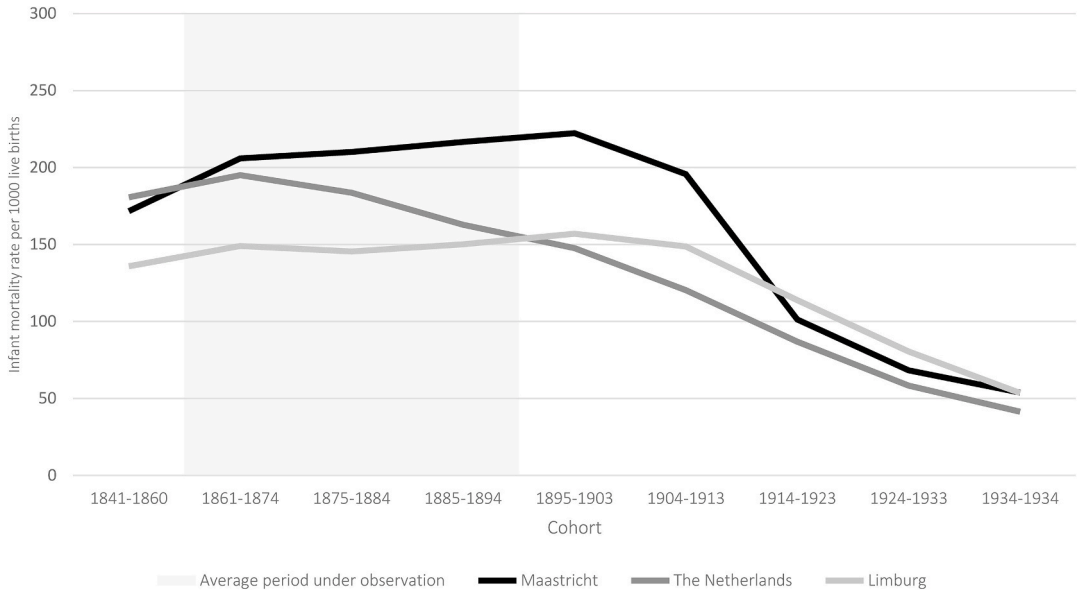


Fig. 1. Infant mortality rate in Maastricht, Limburg and the Netherlands, 1841–1934. Source: Ekamper and van Poppel (2008).

rest of the Netherlands. In Limburg, the IMR was slightly lower than the Dutch average, but remained very stable during our research period. Moreover, between 1875 and 1900, 46% of deaths were children under the age of five (Center for the Social History of Limburg, 2019).

As a consequence of these poor environmental conditions, children who survived until adulthood may have been scarred, resulting in earlier death in adulthood. However, high childhood mortality might have selectively culled the weakest children, resulting in children who survived to adulthood having longer life expectancies (Quaranta, 2014). Given how high Maastricht's early-life mortality rates were, a selection effect in Maastricht during this period likely predominated (Bozzoli et al., 2009).

Against this backdrop, we expect that environmental conditions have a large effect on height, particularly late-adolescent height and growth. We expect the influence of shared family inheritance (a mix of genes and shared environment in early-life) to be comparatively limited. We test this in the first part of our analyses (section 3), using Pearson's correlation and multi-level models. We also expect that those who reach the tallest adult heights fastest to die later than their peers, although this may be muted by scarring. We test this in the second part of our analyses (section 4), using survival models examining all-cause and cause-specific mortality. First, however, we discuss the data used to test these hypotheses (section 2).

2. Data and sample characteristics

2.1. Data

To understand the causes and consequences of height and growth in a pre-transition regime, we exploit a unique dataset, based on a combination of three sources: standing militia (*schutterij)* registers, conscription registers, and the Maastricht Death and Disease Database (MDDD). Table 1 reports the various links among the data sources, and details how we arrive at our sample of 1116 research persons (RPs), including 818 RPs who survive until adulthood (age 35) and with complete covariate information.

We start from Maastricht's *schutterij* registers, which began in 1868 and provide measurements of adult height. All men aged 25 through 34 and resident in Maastricht had to report for the draft. We use the 1868 register because it contains heights and links to the MDDD, the only extant register to do so. Our sample contains virtually all men drafted for the *schutterij* who were born between 1834 and 1843, and who lived in Maastricht in 1868. This yields an initial sample of 1656 RPs with 1482 adult heights.

Table 1
Record linkages.

Schutterij records:	1656
No height in schutterij records	−174
No height in conscription records/no link possible	−366
Dataset with two heights:	1116
No death age	−198
No cause of death	−56
Death before age 35	−44
Dataset with two heights and complete mortality information	818

Sources: Centraal Bureau voor de Genealogie (2019); Centre for the Social History of Limburg (2019); Gemeentebestuur van Maastricht (n.d.); Provinciaal bestuur van Limburg (n.d.)

Next, we link these RPs to their conscription records. We do so by hand on the basis of name, birthplace and birthdate. Conscription occurred at age 19 or 20. The process of Dutch military conscription was nearly identical to that of *schutterij* conscription, with the records containing reliable adolescent height measurements (Quanjer and Kok, 2020). Linking these two sources gives us two height measurements: one from the *schutterij* examination (adulthood), and one at military conscription (late adolescence), along with life-course information. After linking these sources, 1116 RPs remain, with two height measurements and complete covariate information.

Finally, we link the dataset containing RPs' *schutterij* and conscription information to the MDDD. We do so digitally using Levenshtein distances, based on the RP's and his parents' names, along with birth year (Levenshtein, 1966). The MDDD includes all deaths in Maastricht between 1864 and 1955, and contains information on age at death and cause of death. A contemporary physician confirmed and registered individual causes of death. Given that medical knowledge was extremely limited relative to today, there are reliability issues with this data (Reid et al., 2015). Still, a benefit of our dataset is that the causes of death are listed as standalone diseases, versus being contemporaneously grouped by doctors or clerks. This means that we may now classify causes of death according to current medical knowledge. In our dataset, the causes of death are coded in line with the ICD10h (under construction), which is a historical adaptation of the ICD10 (World Health Organization, 2006).

For the cause-specific mortality models, we classify cause of death as

either infectious disease (e.g. tuberculosis), non-infectious disease (e.g. cardiovascular disease), or other. The other category includes violent deaths (e.g. drowning), unclear causes of death (e.g. wasting), or unknown causes of death.

From WieWasWie, a Dutch genealogical website, we locate an additional 56 death certificates of RPs who died outside of Maastricht, but these only contain a death age, not a cause of death (Centraal Bureau voor de Genealogie, 2019). We categorize these RPs' causes of death as unknown. This brings us to a total of 920 RPs with conscription height, *schutterij* height, and death age information. From linking of the *schutterij*, conscription records and death database (with age at death and cause of death), we have a sample of 864 RPs. However, because some RPs died before other RPs enter the analyses and are at risk of dying, we exclude RPs who died before age 35. This brings us to a final sample of 818 RPs for the second part of our analyses.

2.2. Variables

Height and prolonged growth are central to both parts of our analyses. In the first part of our analyses, we use late-adolescent height, adult height, and prolonged growth as continuous outcomes in cm. We proxy prolonged growth trajectory by subtracting late-adolescent height from adult height. In the second part of our analyses, in which height and growth are our key predictors, we use categorical variables to characterize them simultaneously. We initially tested all analyses with height and growth individually, but did not find significant results. These are available on request. Instead, we hypothesize that a combination of height and growth may better-explain mortality. We also use height and growth as combined categorical variables because we expect growth to differently impact mortality at different heights, and because the relationship between growth and height is not linear (see Fig. 2). We use four categories: short-fast (having below-average adult height, and experiencing a below-average amount of prolonged growth); short-slow (below-average adult height, and above-average prolonged growth); tall-fast (above-average adult height, and below-average prolonged growth); tall-slow (above-average adult height, and above-average prolonged growth). We refer to these collectively as 'height groups'. Because we expect that RPs in the tall-fast group to be the longest-lived, we use this group as our reference category. Fig. 2 illustrates these relationships.

Fig. 3 shows how we model the relationships among early-life conditions, height and growth, and mortality. In the first part of our analyses, we look at the direct effects of early-life conditions on height and growth. In the second part of our analyses, we examine the associations of height and prolonged growth on mortality. Our outcome variable is age at death, which is further categorized by cause of death.

We include early-life conditions in our analyses, as they have been found to impact height and mortality (e.g. Quanjer and Kok, 2019; Steckel, 2009; Quaranta, 2014). In the first part of our analyses, we test the association of early-life conditions with height and growth. In the second part of our analyses, we control for early-life conditions, as we assume that they confound the relationship between the height groups and mortality. In doing so, we attempt to capture the direct effect of height groups on mortality. Since we lack detailed early-life information, we proxy early-life conditions with several covariates. First, we use father's occupational class at RP's conscription, and treat this as an indicator of available household resources. We code occupations using HISCLASS5, a widely-used and validated historical occupational classification system (van Leeuwen and Maas, 2011). We anticipate that being from a higher occupational class is associated with taller height, faster growth and later mortality.

Another covariate is birthplace, which is categorized as being born in urban Maastricht, the rural province of Limburg, elsewhere in the Netherlands, or abroad (Belgium or Germany). Being born in urban environments has been found to negatively impact height (Drukker and Tassenaar, 1997). Also, migrants to cities tend to be healthier and live longer relative to those born there (Spitzer and Zimran, 2018). We therefore anticipate that being born outside of Maastricht, and being born in the Limburgian countryside in particular, is positively associated with taller height, faster growth and later mortality.

Also, there is evidence that birth year could impact the relationships between early-life conditions, height and growth, and mortality. We anticipate cohort effects due to the various food crises and epidemics in RPs' early lives. We expect that those who were not very young or were not experiencing their pubertal growth spurt around crisis years (e.g. the 1846–1847 potato famine) are taller, grow faster, and live longer. After

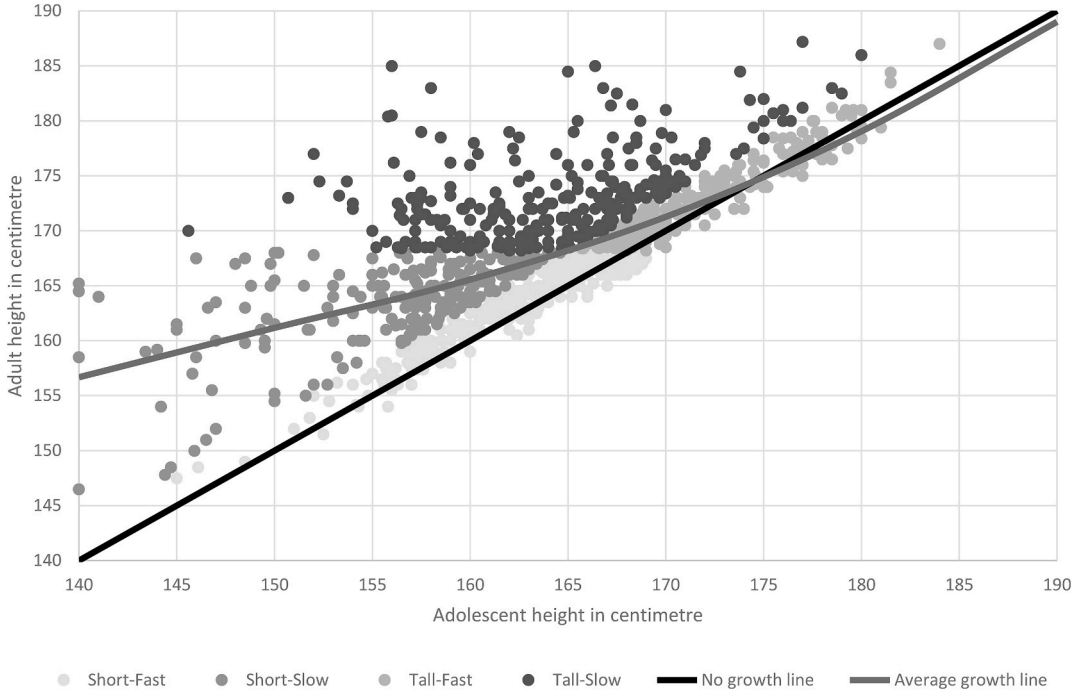
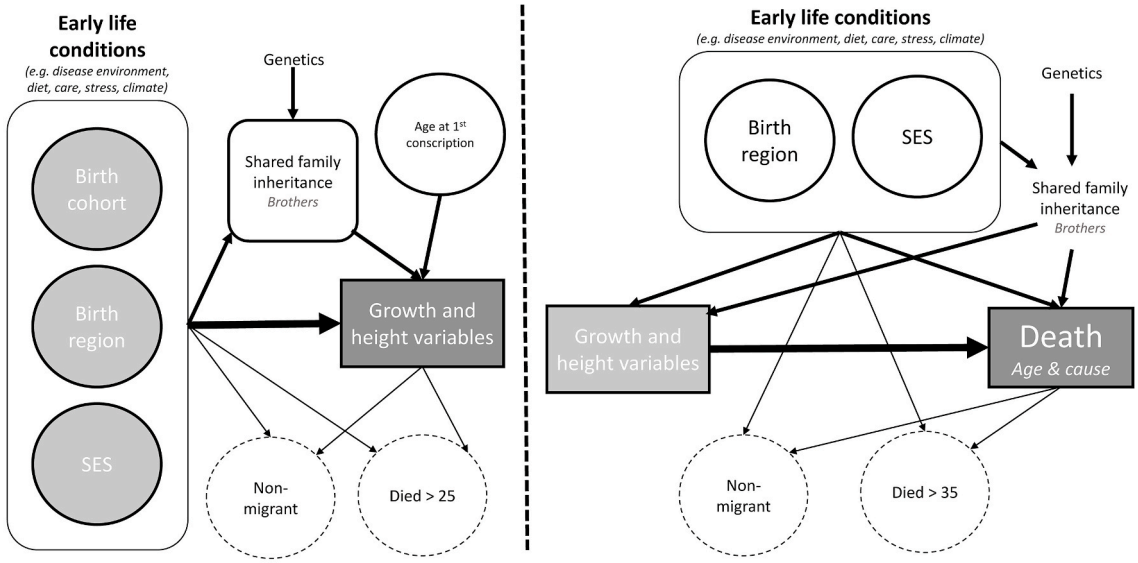


Fig. 2. Growth line between late-adolescent and adult height. Sources: Gemeentebestuur Maastricht (n.d.), Provinciaal bestuur van Limburg (n.d.).



On the left the model testing early life conditions on height and growth. On the right testing the direct effect of height and growth on death. The exposure variables are light grey, the outcome is dark grey. Variables for which we actively adjusted have solid outlines. Variables for which we adjusted by variable selection are white with a dashed outline (see section 4.3). Variables we adjust for by a multilevel model are in a rounded square. The effect we are interested in is depicted by the larger arrow.

Fig. 3. Directed Acyclic Graph (DAG) visualizing the relation between variables in the models used.

an initial test for confounding, we find that birth year does not confound the relationship between the height groups and mortality, and so is only included in the first part of our analyses.

Additionally, we include birth month in the first part of our analyses. We expect that those born earlier in the same year are taller at conscription, because they had more time to grow. Since age at conscription does not confound the relationship between adult height and mortality, we do not include it in the second part of our analyses. Also due to a change in conscription law in 1861, the cohort of 1843 was measured at age 20, while all other cohorts are measured at age 19. To compensate for this extra year of growth, we subtract 1.5 cm from the RPs in the 1843 birth cohorts' conscription heights for both parts of our analyses (Baten and Blum, 2012).

Finally, in the DAG, genetics are included as an unobserved variable. We expect genetics to strongly influence both height and growth, and mortality. We partly control for genetics by using a multi-level model, which accounts for shared family inheritance, in the first part of our analysis. In the second part of our analyses, given that our sample is smaller, we lack the power to use a multi-level model.

2.3. Sample characteristics

Table 2 presents our sample's characteristics. In terms of mortality variables, the average age at death is 59.8 years, with RPs dying of infectious diseases dying earlier on average (53.4 years) than RPs dying of non-infectious diseases (64.7 years). In terms of height, the average late-adolescent height is 163.4 cm, while the average adult height is 167.8 cm. Average growth is 4.4 cm, with a strong right skew. In terms of the height groups used in the second part of our analyses, we see that, among RPs who are below-average adult height, 29% experience a below-average amount of prolonged growth and 23% experience above-average prolonged growth. Among those who are tall, 22% experience a below-average amount of prolonged growth, while 26% experience an above-average amount of prolonged growth (Fig. 2).

Regarding additional covariates, RPs are relatively evenly spread across ten birth years, although there are slightly more RPs from birth years 1838 onward. For SES, a majority of RPs are skilled workers (45.3%) or unskilled workers (31.2%), followed by middle class (10%), farmers (5.7%) and elite (2.4%) during late-adolescence. An additional 5.4% have unknown or no occupations. For birthplace, most RPs are born in Maastricht (77.5%). This is followed by 14.7% from Limburg outside Maastricht, 5.2% from the Netherlands outside Limburg, and 2.4% from abroad.

3. The early-life determinants of height and growth

3.1. Methods

To answer our first research question, we attempt to determine the covariates associated with adolescent height, adult height and prolonged growth. As we have 135 brother groups (containing 2–5 brothers), we exploit this information to test whether height is associated with shared family inheritance (a mix of genetic heritability and contextual factors within the family home), or external environmental factors. First, we test the correlation among brothers groups' late-adolescent heights, adult heights and prolonged growth with Pearson's correlation tests (Alter and Oris, 2008). These correlations vary by time period, birth interval among brothers, and urban or rural context. For late-adolescent height, Alter and Oris (2008) found an average correlation of 49% for brothers conscripted in nineteenth century Belgium, with average birth intervals similar to our study's. Although Alter and Oris (2008)'s context was a mix between rural and urban, and perhaps not directly analogous to ours, we use this 49% as the baseline against which to compare our own findings, as it is the best-available comparison. Based on the Pearson's correlations, we also assess if there are differences in how early-life conditions affect the correlations of conscription height, adult height and prolonged growth, holding shared family inheritance constant.

Next, again exploiting the brother groups, we perform multilevel linear models with random intercepts. We do so to examine the association of early-life conditions (accounting for shared family inheritance) with late-adolescent height, adult height and prolonged growth, with one model per outcome (Feaster et al., 2011). Some of our cases (948 RPs, 135 groups) are clustered at the parental level, so we include a term for the parents (Snijders and Bosker, 2012). Doing so allows us to account for both within- and between-brother group variance. Still, we exploit the full sample of RPs in these models, including those without a brother (who form parental groups of 1). The models are specified:

Table 2
Sample characteristics.

	Obs.	Mean	SD	Min.	Max.
Mortality					
Age at death	818	61.4	14.2	35	95
Noninfectious disease	389	65.4	13.2	35	95
Infectious disease	344	56.1	13.6	35	93
Other/unknown death	85	64.9	14.0	35	86
Height					
Late-adolescent height	1116	163.4	7.4	135.1	190.3
Adult height	1116	167.7	6.5	138.0	189.0
Amount of growth	1116	4.3	4.6	−2.0	29.0
Short adult height, below-average growth (short-fast)[a]	320	162.7	4.1	138.0	168.0
Short adult height, above-average growth (short-slow)[a]	260	163.1	4.2	143.5	168.0
Tall adult height, below-average growth (tall-fast)[a]	249	172.9	4.2	168.2	189.0
Tall adult height, above-average growth (tall-slow)[a]	287	173.2	4.0	168.2	187.2
Additional covariates					
Birth month	1116	6.3	3.5	1	12
HISCLASS5 score[a]:					
Elite	27	168.8	6.8	155.0	182.0
Middle class	111	169.7	6.2	152.0	181.5
Skilled worker	507	167.1	6.8	138.8	189.0
Farmer	64	169.1	6.9	143.5	181.4
Unskilled worker	348	167.6	5.9	146.5	187.0
Unknown	59	168.8	7.1	148.5	184.5
Birthplace[a]:					
Maastricht	867	167.6	6.5	146.5	189.5
Limburg	164	168.3	6.7	138.0	182.5
Netherlands	58	169.8	6.2	159.2	187.2
Abroad	27	165.8	7.2	155.0	181.0
Birth year[a]:					
1834	88	169.2	6.6	155.0	187.2
1835	111	168.0	6.2	147.5	186.0
1836	85	165.7	7.3	148.0	189.0
1837	89	167.4	7.7	143.5	184.5
1838	121	167.2	6.4	149.0	184.4
1839	131	168.3	6.5	154.0	187.0
1840	140	167.7	6.4	138.0	185.0
1841	124	168.1	6.7	147.8	181.4
1842	114	168.4	5.7	156.0	181.5
1843	113	167.5	6.0	154.0	185.0

[a] Mean, SD, min, max based on adult height. See Table 1 for discrepancies in the various totals.

$$Y_{ij} = \beta_{0j} + \beta_1 Birthmonth_{ij} + \beta_2 Occupation_{ij} + \ldots + \beta_7 Occupation_{ij} + \beta_8 Birthplace_{ij} + \ldots + \beta_{11} Birthplace_{ij} + \beta_{12} Year_{ij} + \varepsilon_{ij}$$

$$\beta_{0j} = \beta_0 + u_{0j}$$

where Y represents one of the dependent variables: conscription height, adult height and amount of growth after conscription. We estimate the effect β on Y with all relevant covariates specified in section 2.2 for *i* per brother group *j*.

3.2. Results

We present the brother groups' correlations for late-adolescent height, adult height and prolonged growth in Table 3. The late-adolescent height correlation (0.456) is roughly equivalent to Alter and Oris (2008)'s 0.49. We also find that the correlation of adult height among brothers (0.526) is slightly higher than the late-adolescent height correlation among brothers (0.456). In contrast, the prolonged growth correlation (0.29) is much weaker than that for late-adolescent or adult height.

Table 3
Correlations among brother groups.

	Correlation
Late-adolescent height	0.456***
Adult height	0.526***
Prolonged growth	0.290***

*p ≤ 0.10, **p ≤ 0.05, ***p ≤ 0.01.

Table 4 shows the estimates of early-life environmental conditions' association with adolescent height, adult height and prolonged growth. Father's occupational class is significantly associated with adolescent height and prolonged growth. This relationship to adult height is much weaker, with only RPs in the middle classes being significantly taller than unskilled workers. Regarding birthplace, we do not find significant relationships, except for being born elsewhere in the Netherlands: this is positively, significantly associated with prolonged growth. In terms of birth year, we find that certain birth cohorts (1836 & 1838) are shorter in both late-adolescence and adulthood, but there are no significant differences among birth cohorts based on prolonged growth. Finally, we find that birth month is significantly, positively associated with late-adolescent height and prolonged growth, although it does not have a significant relationship to adult height.

Table 4
Early-life conditions' association with late-adolescent height, adult height and prolonged growth.

	Late-adolescent height β-coefficient	Adult height β-coefficient	Prolonged growth β-coefficient
Birth month	0.217***	0.001	−0.213***
	(0.06)	(0.05)	(0.04)
HISCLASS5 score:			
Elite	3.139*	0.775	−2.346**
	(1.44)*	(1.29)	(0.91)
Middle class	4.959***	2.090***	−2.955***
	(0.80)	(0.72)	(0.50)
Skilled workers	0.971*	−0.258	−1.275***
	(0.52)	(0.46)	(0.32)
Farmers	2.007*	1.029	−1.065*
	(1.02)	(0.91)	(0.64)
Unskilled workers	ref.	ref.	ref.
No/unknown	3.138***	1.128	−2.017***
	(1.05)	(0.93)	(0.66)
Birthplace:			
Maastricht	ref.	ref.	ref.
Limburg	0.780	0.433	−0.352
	(0.65)	(0.58)	(0.40)
Netherlands	0.355	1.922**	1.540**
	(1.01)	(0.90)	(0.62)
Abroad	−0.232	−1.565	−1.289
	(1.46)	(1.29)	(0.90)
Birth year:			
1834	ref.	ref.	ref.
1835	−1.785*	−1.114	0.651
	(1.00)	(0.89)	(0.63)
1836	−2.707**	−3.213***	−0.519 (0.68)
	(1.07)	(0.95)	
1837	−1.031 (1.04)	−1.648*	−0.628
		(0.93)	(0.66)
1838	−1.947**	−1.735**	0.232
	(0.98)	(0.87)	(0.62)
1839	−0.893	−0.648	0.131
	(0.95)	(0.84)	(0.60)
1840	−1.634*	−0.970	0.633
	(0.95)	(0.85)	(0.60)
1841	−0.562	−1.118	−0.581
	(0.97)	(0.87)	(0.62)
1842	−0.971	−0.653	0.296
	(0.99)	(0.88)	(0.63)
1843	−1.490	−2.032**	−0.487
	(1.01)	(0.90)	(0.64)
Adjusted R^2	0.061	0.037	0.078

*p ≤ 0.10, **p ≤ 0.05, ***p ≤ 0.01; Robust standard errors are included in parentheses.

3.3. Interpretation

Our results for the causes of height and growth align closely with our expectations. The 'usual suspects' that contribute to growth trajectory – particularly occupational class – are positively associated with taller conscription height and faster rates of growth. We also find that Dutch migrants into Maastricht are significantly taller. This may be due to occupational class: a quarter of RPs who migrated from elsewhere in the Netherlands are elite or middle class; for other RPs, the share in the elite or middle classes is roughly 10%. It could also be a healthy-migrant effect, whereby migrants tend to be healthier than those who are native-born (Spitzer and Zimran, 2018).

We find some cohort effects as well. Those born in years 1835 through 1838 are shorter in late-adolescence and adulthood, but do not appear to experience significantly longer growth trajectories. These RPs simply do not grow as tall as RPs born in other cohorts. This might be caused by food shortages due to the Crimean War (1853–1856; when these RPs were in late-adolescence). Food shortages may have impacted these RPs in crucial stages of growth (Knibbe, 2007). This is in line with Depauw and Oxley (2019)'s argument that high food prices (and therefore less food) during late-adolescence negatively affect adult height.

We also analyze the contribution of shared family inheritance to height and growth, in a close replication of Alter and Oris (2008)'s methods. The authors used heights from nineteenth century eastern Belgian conscripts, just across the border from Maastricht. For late-adolescent height, we find that shared family inheritance explains 46%, which is roughly equivalent to Alter and Oris (2008)'s 49%. Our study also offers an important elaboration of this paper, which considers conscription height to be terminal adult height. As our results show, growth after late-adolescence (into men's twenties) was not unusual. We find that, while environmental factors are more important during adolescence, their influence lessens by adulthood, with the brother-height correlation increasing to 56% for adult height. This is supported by the comparatively weak correlation (29%) for prolonged growth. We interpret this 29% as shared family inheritance playing a relatively minimal role in determining prolonged growth.

4. The association of height and prolonged growth with mortality

4.1. Methods

To test the association of height and prolonged growth on mortality, we run survival analyses, for all-cause and cause-specific mortality (infectious and non-infectious disease). Survival analyses calculate the expected duration of time until a certain failure event occurs (Cox, 1972). We first run crude and adjusted Cox proportional hazard models for all-cause mortality. We also stratify these models by age at death, because we expect the predictors of death to vary at different ages. We choose age 65 as our cut-off point. While age 65 is above our sample's mean age at death (age 60), it is a widely-accepted marker of old age, and an age after which non-infectious diseases would be more prevalent (Mørkedal et al., 2011). Stratifying by age at death allows us to test whether growth differently impacts the hazard of death at different ages.

In terms of the set-up of the all-cause mortality Cox regressions, age at entry is 35 years, the oldest age at which RPs were conscripted for the *schutterij*. The failure event is death. RPs without age at death information (198 RPs) automatically drop out of the models, because their age at last observation is their age at entry (*schutterij* measurement). For the stratified model of RPs dying before age 65, age at entry is also 35. For the stratified model of RPs who died at age 65 or later, age at entry is 65. We estimate the models:

$$h(t) = h_0(t) \times exp(\beta_1 HeightGroup_1 + \ldots + \beta_4 HeightGroup_4 + \beta_5 Occupation_5 + \ldots + \beta_{10} Occupation_{10} + \beta_{11} Birthplace_{11} + \ldots + \beta_{14} Birthplace_{14})$$

where **h(t)**= the hazard of death; and **βs** = covariates specified in section 2.2.

For the cause-specific mortality analyses, we run dependent competing risk models. With Cox models, the hazard function is always equivalent to the likelihood of dying earlier (Austin et al., 2016). But for cause-specific mortality models, this is not reflective of reality: individuals may die from multiple events, with the occurrence of one event precluding the possibility of others. Dependent competing risk models allow us to analyze cause-specific mortality, while accounting for other possible failure events at the same time (Austin et al., 2016).

We specify two types of competing risks: one in which dying of infectious diseases is the failure event, and one in which noninfectious death is the failure event. We choose these characterizations of cause of death because our small sample size does not allow for any further stratification. There is also strong theoretical grounding for these two characterizations, based on the main causes of death during the epidemiological transition (Omran, 1971). In the case of the infectious model, competing cases are non-infectious death and other causes of death. As with the all-cause mortality models, age at entry is 35. For both infectious and non-infectious death, we run unadjusted and adjusted models. To estimate these models, we use Fine and Gray (1999)'s methods:

$$\tilde{\lambda} j(t|Z(t)) = \tilde{\lambda} 0 j(t) \exp\{\beta' j Z(t)\},$$

where **λ** represents the hazard of death; **t** represents the time of death; **Z** represents cause of death; **j** represents competing causes of death; and **β** represents other covariates specified in section 2.2.

In all survival models, we report hazard ratios (HRs), whereby a chance of dying earlier is compared to the reference group (above-average late-adolescent height RPs who experienced an above-average amount of growth) (Cox, 1972).

4.2. Results

We first explore the associations of the height groups, based on adult height and prolonged growth, with all-cause mortality. Table 5 presents the median life expectancy by height and growth category, across different age groups. We can see that the tall-fast group has a constant lower median life expectancy compared with all other groups.

Next, Table 6 presents the results of our all-cause Cox regressions, including the all-ages models, and those stratified by age at death. In the unadjusted all-ages model, we find that being in the short-fast, short-slow and tall-slow groups is associated with lower hazards of death than the reference group (tall-fast), although this is not significant for any of the height groups. In the adjusted all-ages model, we find similar trends, but here being in the short-slow group and the tall-slow groups is modestly but significantly associated with lower hazards of earlier deaths (both at $\alpha = .10$), relative to the reference (tall-fast) group. For the short-slow group, the hazard of death is 16.8% lower than the tall-fast group. For the tall-slow group, the hazard of death is 16.0% lower.

Turning to the age-stratified models, we do not find any significant

Table 5
Median life expectancies (LE) for height groups, stratified by age groups.

Height groups	Age Groups											
	All ages		40+		50+		60+		70+		80+	
	LE	Obs	LE	Obs	LE	Obs	LE	Obs	LE	Obs	LE	Obs
Short-Fast	62.0	237	64.0	205	68.0	170	72.0	121	78.0	71	85.0	23
Short-Slow	62.0	217	66.0	185	68.0	153	72.0	113	77.0	67	84.0	20
Tall-Fast	58.0	175	63.0	146	65.0	121	70.0	80	76.0	37	87.5	8
Tall-Slow	61.5	236	64.0	212	68.0	167	71.0	127	77.0	68	84.0	20
Total	61.0	865	64.0	748	68.0	611	71.0	441	77.0	243	85.0	71

Table 6
Height groups' association with the hazard of death, all-cause mortality.

	Full sample		Died before age 65		Died at age 65 or later	
	Unadjusted HR	Adjusted HR	Unadjusted HR	Adjusted HR	Unadjusted HR	Adjusted HR
Short-fast	0.8726	0.8742	1.1285	1.1285	0.8267*	0.8021
	(0.08945)	(0.0907)	(0.1535)	(0.1535)	(0.1098)	(0.1045)
Short-slow	0.8818	0.8325*	1.09629	1.0999	0.9320	0.9721
	(0.0931)	(0.0905)	(0.1541)	(0.1570)	(0.1500)	(0.1572)
Tall-fast	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
Tall-slow	0.8816	0.8404*	1.0223	0.9904	0.9134	0.9375
	(0.0913)	(0.0894)	(0.1396)	(0.1379)	(0.1457)	(0.1949)
N failure events	818	818	450	450	368	368

*p ≤ 0.10, **p ≤ 0.05, ***p ≤ 0.01; Robust standard errors are included in parentheses.

Table 7
Height groups' associations with the hazard of death, cause-specific mortality.

	Infectious death		Non-infectious death	
	Unadjusted HR	Adjusted HR	Unadjusted HR	Adjusted HR
Short-fast	0.9211	0.8994	0.9024	0.8741
	(0.1452)	(0.1459)	(0.1358)	(0.1339)
Short-slow	1.0784	0.9678	0.8674	0.8807
	(0.1712)	(0.1591)	(0.1359)	(0.1401)
Tall-fast	Ref.	Ref.	Ref.	Ref.
Tall-slow	0.9711	0.8755	1.0007	1.0537
	(0.1548)	(0.1488)	(0.1475)	(0.1595)
N failure events	344	344	389	389

*p ≤ 0.10, **p ≤ 0.05, ***p ≤ 0.01; Robust standard errors are included in parentheses.

associations between the height groups and mortality for those who die below the age of 65. However, we find that, among RPs who died at age 65 or older, being in the short-fast group is modestly but significantly associated with a lower hazard of death relative to the reference group, in both the unadjusted and adjusted models. In the unadjusted model, this is associated with a 17.3% lower hazard than the tall-fast reference group, and is significant at $\alpha = 0.10$. This is no longer significant in the adjusted model.

Table 7 presents the results of our cause-specific mortality models. Here, we find no significant association of the height groups and the hazard of death, in the models for death from infectious and non-infectious diseases. This is highly insignificant for all of the height groups.

Ultimately, we find some evidence that experiencing a shorter growth trajectory and being taller in adulthood (tall-fast) is associated with earlier death: in the adjusted all-cause mortality model, being taller and reaching terminal adult height over a shorter period is significantly (at a = 0.10) associated with an increased hazard of death. For the cause-specific mortality models, we find scant evidence that the height groups are significant predictors of dying of non-infectious or infectious disease.

4.3. Interpretation

Our initial hypothesis - that the faster growing, taller height group would be associated with a lower cause of death – is in line with existing studies on the relationship between height and mortality (e.g. Waaler, 1984; Costa, 2004). However, we do not find significant relationships between adult height and mortality, and, when we take into account growth trajectory, we find that this tall-fast group has the highest hazard of mortality. There may be practical explanations for this. For example, we do not have access to weight information. This may be why these studies, looking at BMI's relationship to mortality, find clearer results. Still our results for the all-cause mortality models raise the question: why would the hypothesized long-lived tall-fast group be associated with a higher mortality hazard?

An explanation of these different later-life mortality hazards might be a difference in selection in early life (Quaranta, 2014). Bozzoli et al. (2009) argued that, when environmental conditions are sufficiently poor and mortality is sufficiently high, a strong selection mechanism may be at play, with the survivors being fitter than the non-survivors, as well as those who did not experience this selection. The tall-fast group's growth trajectory is most similar to growth trajectories in contemporary, low mortality populations, with individuals reaching taller terminal heights at earlier ages. It appears that the tall-fast group was relatively shielded from harmful early-life conditions, relative to their peers. As a result, this group was less strongly selected in early-life and, consequently, is weaker overall in adulthood. This may have resulted in the observed excess mortality in later-life. It may be because we are looking at a sample different from Waaler (1984) and Costa (2004), one that likely encountered more food shortages and epidemics, that we find these surprising results.

The findings from the cause-specific models are less clear. This might be the result of a power problem and/or the creation of broad, aggregated categories. However, it may also be an accurate picture of the influence of height on cause-specific mortality. Diseases that have been shown to be impacted by height, particularly cancer, are less prevalent in pre-transition populations (Omran, 1971; Davey Smith et al., 2000).

In sum, we surprisingly find a higher mortality hazard for the tall-fast group, whom we initially predicted to be the longest-lived. We also find evidence that the relationship between height, growth and mortality is

much less straightforward in our sample than in those from studies conducted in lower-mortality settings. We therefore have made an important first step to understanding how the relationship between growth and mortality may vary by context. Our results present a new puzzle: do our findings reflect the true height-mortality relationships present in late-nineteenth century Maastricht, or do we have data problems polluting our results?

We first examine the possibility that data problems may be impacting our results. As mentioned, our sample size is relatively small. Particularly, our cause-specific mortality models, with only a few hundred cases per sub-group, may be suffering from a straightforward power problem and an accordant Type II error. For future studies, larger samples could yield more accurate measures.

However, this is only useful if the underlying population is unbiased. In our study, measurement error may have biased our sample, particularly with our two height measurements. Our sample contains some shrinking: a small share of our RPs display shrinking of more than 2 cm between conscription and their examination for the *schutterij*. We have deleted these observations from our data, because they likely are the result of writing or transcription errors. We keep those who shrunk fewer than 2 cm for two reasons. First, this difference is possible, depending on time of day: young adults have been found to be, on average, 1.1 cm taller in the morning than in the evening, due to spinal compression over the course of the day (Nixon, 1986). Second, while transcription errors resulting in RPs shrinking are obvious, errors in the other direction (i.e. overstating RPs' actual heights) are undetected. Including negative values of growth may balance out these positive errors.

Further, we may have collider biases, or endogenous selection biases, in our sample (Elwert and Winship, 2014; Schneider, 2020). Our sample is comprised only of those who survived until adulthood (*schutterij* measurement), and only of those who died in Maastricht (in other words, non-migrant adult survivors) (see Fig. 3). This is problematic, because out-migration and early death are related to early-life conditions, height and growth, and mortality. It may be that we are detecting a selection bias, rather than a selection effect. In the case of out-migration, we attempted to mitigate this problem by finding death certificates outside of Maastricht. We also test whether SES and migration had an effect on mortality outcomes, and find no significant changes in our results. This raises our confidence that we are finding a true selection effect.

Overall, we believe that the effects we have detected on mortality are reliable, particularly for the causes of adult height and the prolonged growth trajectory and its consequences for all-cause mortality. We find preliminary evidence that the relationship between growth and height on the one hand, and mortality on the other is not as straightforward in this pre-transition regime as appears to be in post-transition ones. This echoes Sear (2010), a literature review that showed that height's positive relationship to longevity was only found in healthy Western European populations. Our study is a preliminary step to better-understanding these relationships in different contexts.

5. Conclusion

With this paper, we add an important perspective to the study of height, growth and mortality. We look at both height and prolonged growth, and explore both their causes and mortality consequences. We exploit a unique dataset of men who lived and died in the nineteenth century Dutch city of Maastricht. This sample stands out for its life-course information, two height measurements, and specific causes of death in a pre-transition regime. Further, to our knowledge, ours is the first study to examine the relationship between prolonged growth and mortality in a population prior to the epidemiological transition. Most related studies explore the influence of adult height on mortality, and do so in post-transition contexts (Silventoinen et al., 1999; Davey Smith et al., 2000; Waaler, 1984). As mentioned, our population has a life-expectancy similar to countries with the lowest life-expectancies today. Also, a number of contemporary low-income African and South/South-East Asian countries have not yet experienced secular growth trends and appear to be mid-health transition, much like the Netherlands in this period (NCD Risk Factor Collaboration, 2016). Our study may therefore shed some light on the relationship between height and mortality in modern-day, pre-transition populations.

In terms of the causes of height and prolonged growth, we find that external environmental factors, relative to shared family inheritance, plays a larger role in growth and height during adolescence. However, shared family inheritance is more strongly associated with terminal adult height. In terms of the consequences of adult height and prolonged growth, we surprisingly find that the tall and fast growers – whom we expected to be the longest-lived group - are the shortest-lived, presumably due to less severe selection in early life. More weak tall and fast growers may have survived until adulthood, and therefore died earlier relative to other adults. This raises the question of how selection effects mitigate or reverse the relationship between adult height, the growth trajectory and mortality. Our study offers an important step to understanding these relationships outside the healthy, wealthy and tall Western world.

Credit author statement

Kristina Thompson: Conceptualization, Methodology, Formal analysis, Writing - original draft, writing – reviewing/editing, Björn Quanjer: Conceptualization, Methodology, Formal analysis, Writing - original draft, writing – reviewing/editing, Mayra Murkens: Conceptualization, Methodology, Writing - original draft, writing – reviewing/editing.

Acknowledgements

We would like to thank Jan Kok, France Portrait, Willibrord Rutten and George Alter for their comments on early versions of this paper. We would also like to thank the Sociaal Historisch Centrum Limburg for access to the Maastricht Death and Disease Database. We also would like to acknowledge de Nederlandse Organisatie voor Wetenschappelijk Onderzoek for funding the Giants of the Modern World project (project number: 360-53-190).

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